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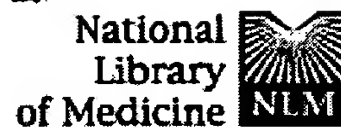
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Proc Natl Acad Sci U S A. 1991 Jul 1;88(13):5528-32.

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The scurfy mouse mutant has previously unrecognized hematological abnormalities and resembles Wiskott-Aldrich syndrome.

Proc Natl Acad Sci U S A. 1990 Apr;87(7):2433-7.

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J Clin Invest. 2001 Jan;107(2):155-7. No abstract available.

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Cellular and molecular characterization of the scurfy mouse mutant.

J Immunol. 1999 Mar 1;162(5):2546-54.

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The C. elegans PTEN homolog, DAF-18, acts in the insulin receptor-like metabolic signaling pathway.

Ogg S, Ruvkun G

Department of Molecular Biology, Massachusetts General Hospital, Boston 02114, USA.

An insulin-like signaling pathway, from the DAF-2 receptor, the AGE-1 phosphoinositide 3-kinase, and the AKT-1/AKT-2 serine/threonine kinases to the DAF-16 Fork head transcription factor, regulates the metabolism, development, and life span of *Caenorhabditis elegans*. Inhibition of *daf-18* gene activity bypasses the normal requirement for AGE-1 and partially bypasses the need for DAF-2 signaling. The suppression of *age-1* mutations by a *daf-18* mutation depends on AKT-1/AKT-2 signaling, showing that DAF-18 acts between AGE-1 and the AKT input to DAF-16 transcriptional regulation. *daf-18* encodes a homolog of the human tumor suppressor PTEN (MMAC1/TEP1), which has 3-phosphatase activity toward phosphatidylinositol 3,4,5-trisphosphate (PIP3). DAF-18 PTEN may normally limit AKT-1 and AKT-2 activation by decreasing PIP3 levels. The action of *daf-18* in this metabolic control pathway suggests that mammalian PTEN may modulate insulin signaling and may be variant in diabetic pedigrees.

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